in the brain regions which have synaptic conjunctions with the primary brain lesion, it also exhibits efficacy for the prevention of the secondary lesions of many neurodegenerative diseases (e.g. Alzheimer's disease, Pick's disease, spinocerebellar degeneration, Parkinson's disease, chorea, polyglutamine diseases, amyotrophic lateral sclerosis or multiple sclerosis), and is expected to improve QOL (Quality of Life) for patients as a result of reducing progression of higher nervous function disorders caused by these diseases. As described in JP98/365560 and PCT/JP99/02550 ("Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1"), the pharmaceutical composition comprising ginsenoside Rb_1 is thought to exhibit efficacy against the primary lesions of these neurodegenerative diseases through its inhibitory effect on apoptosis-like nerve cell death and through upregulation of $Bcl-X_L$ expression.

Further, the intravenous administration of ginsenoside Rb_1 of the present invention markedly ameliorates paralysis of animals with spinal cord injuries. It is well known that the nervous tissues are more vulnerable to trauma than any other peripheral tissues. The fact that the pharmaceutical composition comprising ginsenoside Rb_1 exhibits conspicuously favorable effects for therapy and treatment of spinal cord injuries, indicates that ginsenoside Rb_1 is effective for the

treatment of traumatic injuries to the peripheral tissues as well as the central nervous tissues. As shown in the following example 4, in rats with spinal cord injuries in which compression was loaded to the lower thoracic spinal cord, intravenous administration of ginsenoside Rb_1 ameliorated paralysis of both lower limbs (paraplegia) and enabled the rats to stand up after spinal cord injuries. The rats with spinal cord injuries, to which only physiological saline (i.e. vehicle) was administered, remained paralyzed in both lower limbs and could not stand up. In addition, intravenous administration of Solu-Medol (methylprednisolone), which is used for treatment of spinal cord injuries at present, could not ameliorate paralysis of both lower limbs (paraplegia) in rats with spinal cord injuries. On the basis of these findings, the therapeutic effect of ginsenoside Rb_1 on spinal cord injuries is thought to be the most potent among the compounds so far examined. Consequently, it is expected that, in the future, ginsenoside Rb_1 or its metabolites will be used as a leading compound(s) in the development of various remedies for spinal cord injuries, neurotrauma and traumatic injuries.

Further, a specific feature of the pharmaceutical preparation or composition comprising ginsenoside Rb_1 of the present invention, which should not be overlooked, is the fact that it does not show any adverse effects. For example, as described in JP98/365560 and PCT/JP99/02550 ("Brain cell or

nerve cell-protective agents comprising ginsenoside Rb_1"), even though ginsenoside Rb_1 is added to normal cultured nerve cells or neurons, which are not treated with sodium nitroprusside (SNP), a nitric oxide monoxide donor, it shows no effect on normal metabolic activity. Moreover, ginsenoside Rb_1 at low extracellular concentrations (1 - 100 fg/ml) protects only nerve cells against SNP-induced injuries. Consequently, ginsenoside Rb_1 does not affect normal neuronal functions but can give a favorable effect only on lesion tissue. This point can be emphasized as a superior property of ginsenoside Rb_1 than glutamate receptor antagonists under developing as neuroprotective agents at present.

It has also been reported that no effects of intracerebroventricular administration of ginsenoside Rb_1 on brain temperature, cerebral blood flow and blood pressure are observed (Lim J.-H. et al. Neurosci. Res., 28, 191-200, 1997; Zhang B. et al., J. Stroke Cerebrovasc. Dis., 7, 1-9, 1998). We have confirmed that intravenous infusion of ginsenoside Rb_1 in rats, in a dose of 60 μg/day, does not affect the cerebral blood flow. It is also known that ginsenoside Rb_1 does not promote bleeding tendency. No adverse effects were detected within a range of careful observation on animals, to which ginsenoside Rb_1 of the present invention was administered.

As described in JP98/365560 and PCT/JP99/02550 ("Brain